Modern Environmental Health Hazards: A Public Health Issue of Increasing Significance in Africa

Supplemental Material

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Abbreviations:

Hg: Mercury

MEHH: Modern environmental health hazard

NOx: Nitrogen oxides

PAH: Polycyclic aromatic hydrocarbon

Pb: lead

PM: Particulate matter

ppb: parts per billion

ppm: parts per million

SO_2: Sulfur dioxide

SOx: Sulfur oxides

U.S. EPA: United States Environmental Protection Agency

VOC: Volatile organic compounds

WHO: World Health Organization

μg/m: micrograms per meter

μm: micrometer

Supplemental Material, Appendix A

Indoor Air Pollution

Though indoor air pollution is recognized as a traditional hazard associated with underdevelopment and rural environments, it is also an emerging issue in low income and rapidly urbanizing areas, primarily as a result of limited access to cleaner burning fuels. In Nigeria, the use of less clean fuel alternatives has increased, and erratic supply is cited as one of the reasons why electricity (one of the cleanest form at the household level) is not a popular energy source for cooking (Anozie et al. 2007). One-third of urban households in Nigeria surveyed in this study reported using fuel wood and charcoal for cooking. Another study conducted in a different region of Nigeria also reported a consistent trend away from fossil fuels (which generally burn cleaner) and towards the use of fuel wood (Ogbueh 2006). The author suggested a relationship between the observed trend and the increased cost and scarcity of cleaner fuel sources such as kerosene.

In sub-Saharan Africa, an estimated 77% of the population depend on solid fuels (Rehfuess et al. 2006), and are therefore potentially exposed to air pollution released during the inefficient combustion of these fuels. Typical indoor air pollutants resulting from the burning of wood, biomass, fossil fuels and coal include SOx, NO_X, PAHs, aldehydes such as acrolein, formaldehyde and acetaldehyde, PM and VOCs. Because solid fuels burn inefficiently, high concentrations of these air pollutants typically in excess of health based standards are released during combustion. Mean concentrations of PM less than 10 μm in size (PM_{10}) released from wood, charcoal and coal combustion during the preparation of one meal (approximately 1.5 hours) can range from 540 $\mu g/m^3$ to 1200 $\mu g/m^3$ (Ellegård 1996). Mean PM_{10} emissions from kerosene which is supposed to be a cleaner burning fuel was higher than emissions from charcoal

(760 μg/m^3) in this study. High air toxics emissions from kerosene may be attributable to "poor kerosene quality" (Ogbueh 2006) or inefficient stoves. Because meal preparation is a routine activity in homes, the use of fuels that burn inefficiently is a source of chronic exposure to toxicologically significant levels of related air pollutants particularly for women and their children. The impact of exposure to such high concentrations of air toxics may be more severe in children relative to adults because lung development is an ongoing and vulnerable process throughout childhood (Bearer 1995), as are other developmental processes including those occurring throughout pregnancy.

Other sources of indoor air pollution in African homes include pesticides (Desai et al. 2004) such as sprays, bombs and mosquito coils, and cigarettes. The mosquito coil is recommended and used for repelling mosquitoes in areas with high vector density and high transmission rates for mosquito transmitted diseases such as dengue and dengue hemorrhagic fevers (WHO 2005), and malaria. Though useful in preventing acute vector transmitted diseases, they can emit high concentrations of the chemicals similar to those found in biomass smoke, and in addition pesticides such as pyrethrin. Liu et al. (2003) estimated that the amount of $PM_{2.5}$ emitted from one mosquito coil is comparable to smoking at least 75 cigarettes a day. High concentrations of other chemicals such as aldehydes were also emitted during coil combustion in their study.

The prevalence of cigarette smoking, a well documented source of indoor air toxics, is on the increase among subsets of the African population. In some countries, rates of cigarette consumption have increased dramatically, particularly among the youth and women. Between 1990 and 2000, annual cigarette imports to Nigeria increased from 198 million to 3 billion sticks. Smoking prevalence in adult females aged 15 years and above in the same country experienced a

dramatic increase from 1.7% to 17% over the same time frame (Oluwafemi 2003). Increased acceptance of smoking particularly among females may translate to increased smoking in homes and public places, and therefore increased exposures of the general population and particularly children to environmental tobacco smoke.

Supplemental Material, Appendix B

The Impacts of MEHHs in the African Context

The environmental health challenges created by inadequate recognition and management of MEHHs may exert especially grave impacts on population health and well-being for a number of reasons:

- A large proportion of Africa's population is comprised of children, a sub-population that is vulnerable to environmental health hazards. In some countries, more than 50% of the population is comprised of children (ages 0 to 18 years). Given current projections under assumptions of medium fertility rates, normal mortality (including the impact of AIDS), and normal international migration, one-third of Africa's population will still be accounted for by children ≤14 years by the year 2050 (Department of Economic and Social Affairs 2007). Based on these projections, children will remain a significant proportion of the African population for several decades to come.

  Children are vulnerable to environmental health hazards for several reasons: they have unique behavioral patterns such as hand to mouth behavior that predispose them to exposures to toxic agents in environmental media such as dust or soil (Cohen Hubal et al. 2000); they experience higher exposures to toxics in environmental media because weight for weight they consume more food and water, and breathe more air than adults; they are more vulnerable physiologically because of their developing organs and metabolic systems, and in ways that may enhance the uptake of toxic agents or increase the likelihood of harm; their life stage is one characterized by rapid growth and development; and they have more time to develop chronic diseases (Suk et al. 2003). Given these vulnerabilities, the impacts of unchecked proliferation of MEHHs in terms of

the proportion of the population affected and the extent of the impacts can reasonably be expected to be profound for Africa.

- Many African populations suffer predisposing conditions that may increase the risk of exposure to toxic agents or disease, the severity of disease, or the body's ability to withstand harm or rebound from toxic insults. These factors include poverty, poor social and economic conditions, and lack of access to preventive and curative health care. Most African countries are poor and lagging in the area of human development; the continent hosts 76% of the 50 least developed countries in the world based on indicators of income, human assets, health, nutrition, school enrollment, adult literacy and economic vulnerability (UNCTAD 2005). Poverty is a known risk factor for both exposure to toxic agents and adverse health outcomes, and particularly for children. Children living in poverty for example, are more likely to be employed in child labor and in dangerous professions such as artisanal mining where the risk of exposure to toxics is high (Goldman and Tran 2002). Children living in poverty are also more likely to live in housing conditions that expose them to hazards. In the United States significant Pb-based paint hazards are more prevalent in resource poor homes (Jacobs et al. 2002).

  Poor children are also more likely to have predispositions that enhance exposure to or the effect of toxic substances. For example, poor children are more likely to suffer micronutrient deficiencies (Rivera et al. 2001), which may enhance the uptake of toxics such as heavy metals within the body (Peraza et al. 1998). Pb uptake and absorption for instance is enhanced by calcium, iron, protein and zinc deficiencies (Mahaffey 1981; Mahaffey and Michaelson 1980).

Although predisposing conditions such as micronutrient deficiencies may enhance exposure to or the effects of hazards, many of these conditions also have independent adverse effects on the same target organs or systems, and the aggregate or cumulative effects of these insults are not always well understood. For example, iron deficiency (which enhances the uptake of Pb) is an independent predictor of cognitive deficits (Kordas et al. 2004), an effect that is also associated with Pb exposure. .

- Interactions between modern and traditional environmental health hazards may have adverse consequences on the ability to resist disease. Studies in mining communities in the Americas show that exposure to mercury could impair host resistance to malaria (Silbergeld et al. 2000; Silva et al. 2004). In Canada, increased risks of lower respiratory tract (LRTI) and ear infections have been observed in Inuit children following prenatal exposure to polychlorinated biphenyls (Dallaire et al. 2006). In Africa, both malaria and LRTI are among the top five contributors to the environmentally related burden of disease (Prüss-Üstün and Corvalán 2006), and children bear a disproportionate fraction of this burden.

These findings suggest that MEHHs in the most general terms may exacerbate the disease burden attributable to endemic and poorly managed traditional hazards, or even to other hazards that are not explicitly associated with the environment. These findings also illuminate the possibility that novel risks may evolve from the juxtaposition of modern and traditional hazards. As mentioned earlier, an additional complication of interactions between MEHHs and traditional hazards is that many African communities are significantly burdened by other factors such as conflict, malnutrition, and poor health

including co-existing conditions like HIV/AIDS that also individually or collectively affect the body's ability to resist illness.

- Unlike most traditional hazards, a long latency period from when exposure occurs to clinical manifestation of disease is generally characteristic of many MEHHs (Corvalán et al. 1999), with illness sometimes occurring decades post-exposure. Examples of diseases with long latencies that are associated with MEHH exposure are asbestos related diseases such as lung cancer and mesothelioma, which take decades past exposure on average to manifest. Persons exposed to agents with long latency characteristics in childhood, as may be the case for a sizable proportion of the African population, have enough years of life to experience the adverse impacts of such exposures.
- Like many other hazards, the health consequences of MEHHs not only affect the individual, but also the individual's larger community. We illustrate this concept with the example of Pb. Pb exposure in childhood is associated with IQ deficits (Lanphear et al. 2005). A lowered IQ in childhood adversely impacts educational attainment, and therefore future earning potential (Schwartz 1994). When aggregated across individuals to the community level, such losses in educational attainment and future earning potential can translate to significant economic losses for the community. Researchers in the US predict significant gains (hundreds of billions of dollars) in earning power for recent cohorts of children in the US relative to their counterparts from a generation earlier in the 1970s who had much higher Pb exposures (Grosse et al. 2002). At a minimum, higher earning power for the individual translates into higher tax revenues that are used for societal gain. At this point in time when Africa could maximally utilize all of its human

resources for development, these potential types of subtle yet powerful negative influences on its human resource pool are not desirable.

Additional examples of the potential range of adverse impacts that MEHHs can have on both individual and community well being can be drawn from the emerging literature on the relationship between Pb and criminal or violent behavior. A study by Needleman et al (2002) for example suggests an association between Pb exposure and criminality measured as adjudicated delinquency for acts that include robbery, firearms possession, and assault. Another study by Nevin (2000) correlates temporal changes in blood Pb levels and temporal trends in violent crime in the United States. If as suggested by Nevin (2000), trends in Pb exposure are a significant factor associated with subsequent trends in negative social behaviors such as robbery and assault the total social cost of just one MEHH can be far reaching and significant.

Supplemental Material, Appendix C

Table 1: Blood Levels of Hg and Pb in Sampled African Populations.

	Biological Levels	Percent > Reference Value[k]	Age	Measurement Year	N	Country	References
	(µg/dL)						
Pb	44.4 - 51.5[i,a]	n/a	7y	2002	1	South Africa	Mathee et al. 2003
	12.3 – 20.1[j,b]	69.6	9-60m	-	56	Egypt	Boseila et al. 2004
	6.1±1.8[l,b]	5[d]	8-12y	1998	19	Senegal	Diouf et al. 2003
	23 - 110[m,c]	100	Adults	-	382	South Africa	Robins et al. 1997
	26 – 97[m,c]	100	Adults	-	86	Nigeria	Adeniyi and Anetor 1999
	15 - 63[m,a+b]	100	Adults	-	880	Nigeria	Adeniyi and Anetor 1999
	9 – 27[m,e]	98[f]	6-10y	-	86	South Africa	von Schirnding 2003
	6 – 22[m,e]	85[f]	6-10y	-	68	South Africa	von Schirnding 2003
	(ppb)						
Hg	<LOD[g] – 33.3[5,h]	-	Mixed	2003	252	Tanzania	Appleton et al. 2004
	<LOD[g] – 44.8[5,h]	-	Mixed	2002	180	Ghana	Rambaud et al. 2003
	<LOD[g] – 100.8[5,h]	-	Mixed	2004	269	Zimbabwe	Boese-O'Reilly et al. 2004

Notes:
[a]Urban; [b]Rural; [c]Occupational; [d]Estimated from available data. One out of 19 rural children had a blood Pb level > 10 µg/dL; [e]Study was conducted in mining communities; [f]Percent of sample ≥10 µg/dL; [g]LOD for Hg in blood was 0.2 ppb; [h]Estimated arithmetic mean/median blood Hg values in the studies were: Tanzania (1.7 ppb/2.92 ppb), Ghana (10.7 ppb/10.4 ppb), and Zimbabwe (12.6 ppb/5.62 ppb); [i]Initial screening of child yielded the lower blood Pb level. Screening a month later yielded the higher blood Pb value. Pb paint was suspected as the source of exposure; [j]Range of mean blood Pb levels, where mean is presented as Mean±SD; [k]Reference blood Pb value is 10 µg/dL, and the reference blood Hg value is 5.8 ppb. The blood Hg reference level is approximately equivalent to U.S. EPA's reference dose of 0.1µg/Kg body weight/day. The reference value for blood Hg is based on studies of neurological effects of Hg in populations exposed to organic mercury primarily via seafood ingestion; [l]Mean blood Pb presented as Mean±SD; [m]Range of contaminant levels measured in sampled population.

Table 2: Selected Levels of Pb in Consumer Products in Africa

Consumer Product	Levels (ppm)	Country	Study References	Recommended/ Allowable Limit (ppm)	References
Residential paint	Up to 29000[a]	South Africa	Montgomery and Mathee 2005	600	CPSC 1977
Paint on children's toys	Up to 145000[a]	"	Mathee et al. 2007	"	
For sale pigmented enamel household paint	Up to 189000[a]	"	Mathee et al. 2007	"	
For sale pigmented and white glossy household paint	3615 – 40515[b]	Nigeria	Adebamowo et al. 2007	"	
Crayons	8200 - 10650[c]	South Africa	Okonkwo and Maribe 2004	0.1[d]	FDA 2006
Pencils	79 – 1160[c]	"	Okonkwo and Maribe 2004	"	

[a]Range of Pb concentrations measured in samples; [b]Range of median Pb concentrations across different sampled color pigments; [c]Range of arithmetic mean Pb concentration; [d]Children frequently place these objects in their mouths, and also often engage in hand to mouth behavior that increases the risk of exposure to Pb from these sources. Therefore, the FDA recommended limit for candy is considered an appropriate surrogate benchmark for evaluating exposure to Pb from crayons and pencils.

Table 3: Selected Recent Outdoor Levels of Air Pollutants in African Environments

Air Pollutant	Ambient Levels ($\mu g/m^3$)	Measurement Year	Location Description	Country	Study References	Guideline Value/ Standard[d,e] ($\mu g/m^3$)	References for Guideline Value/Standard
SO_2	127 – 1385 [a,b]	2002-2003	Urban	Tanzania	Jackson 2005	655	ARB 2008
	128 – 2483 [a,b]	2003	Bus Stations	"	Msafiri 2004	"	
	17 -328 [b,c]	2000	Urban	Zimbabwe	Chibanda 2004	50	WHO 2000
	32 -124 [b,c]	2001	"	"	Chibanda 2004	"	
PM_{10}	100 – 250[c]	2000-2002	Urban, Residential	Egypt	Mohamed 2004	20	WHO 2006
	Up to 450 [c]	2000-2002	Industrial Sites	"	Mohamed 2004	"	
NO_2	25 – 83 [c]	2002	Mixed	Egypt	Mohamed 2004	40	WHO 2006
	75 – 83 [c]	2002	Urban	"	Mohamed 2004	"	
	21- 40 [c]	2000-2001	"	Zimbabwe	Chibanda 2004	"	
	88-297 [a,b]	2003	Bus Stations	Tanzania	Msafiri 2004	200	WHO 2006
	18-53 [a]	2002-2003	Urban	"	Jackson 2005	"	

[a]Range of hourly mean concentration across different sites; [b]Reported mean levels at >50% of sites exceed the relevant standard/guideline value; [c]Range of annual mean concentration across different sites; [d]Each guideline value/standard to which an "ambient level" is compared has the same averaging time; [e]Both the SO_2 "Standard" from the California Air Resources Board, and the other reference values which are specifically referred to as WHO "guideline values" are health-based. However, a "standard" is different in a number of ways including that the numerical value is associated with a specific measurement method, and the method for data collection is specified.

Table 4: A Selection of Operational Industries in Africa and their Potential Air Emissions

Industry	Air Emissions
Pulp and Paper	$_2$, NOx, H_2S, mercaptans
Mining (Sectors: metallic ores, heavy metals, non-metallic ores, energy e.g. coal and oil, industrial minerals	SO_2, CO_2, CO, NOx, PAH, dust, PM, heavy metals
Inorganic Chemical Industry	$_2$, SO_2, PM, Pb, fluorine
Organic Chemical Industry	PM, VOCs, H_2S, SO_2, NOx
Metallurgical Industry	Acid mists, PM_{10}, SOx, NOx, fluorides
General Manufacturing and Trade	SO_2, H_2S, NOx, CO_2, hydrogen cyanide, ammonia, VOCs, heavy metals e.g. mercury, vanadium, nickel

Source: Shannon 2004$_{SO}$

NO

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